State of Connecticut
GENERAL ASSEMBLY

COMMISSION ON CHILDREN

Public Health Committee
Public Hearing
March 14, 2007
Testimony Submitted by
Elizabeth C. Brown, Legislative Director

Senator Handley, Representative Sayers and Members of the Committee. My name is Elizabeth C. Brown and I am the Legislative Director for the Commission on Children. I appreciate the opportunity to testify this morning in support of two bills related the healthy development of children.

Support: SB 1220, An Act Concerning the Birth to Three Program-
We ask that you delete Section 2 referring to personnel issues. This issue was discussed last year and the Department is not moving forward with this option.

The Commission is honored to be a member of the Early Childhood Cabinet established to develop a comprehensive plan for children birth to eight. The goal is to ensure every child is "Ready by Five and Fine by Nine" as the title of the Cabinet Report reflects. One of the top priorities identified by the Cabinet is the expansion of the Birth to Three early intervention program administered by the Department of Mental Retardation.

In 2001 during the state budget crisis, the Birth to Program eligibility requirements were dramatically curtailed, eliminating children in high risk categories. These changes resulted in almost 1000 less children served in 2006.

Why is the Birth to Three Program Important- it really works!

- It costs $7000 (net cost to state and federal funding) to serve a child in Birth to Three for 12 months v. $17,000 cost of preschool special education in Connecticut.
- Of the 2,666 children who left Birth to Three at age three in FY 05, 70% were eligible for preschool education. Therefore, 794, children **did not** receive preschool special education for a savings of $12,902,500!

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the important educational components of bill 683 be reviewed in light of this bill and necessary changes be made to ensure that the intent is accurately outlined.

Thank you for your leadership on these important health issues for children.

PEDIATRICS®

Lead Exposure in Children: Prevention, Detection, and Management

Committee on Environmental Health

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of age had a median blood lead concentration of 15 μg/dL.[10] In 1988–1991, the median was 3.6 μg/dL[11]; in 1999, the median was 1.9 μg/dL.[12] Although concentrations have decreased in all children, black children and poor children continue to have higher blood lead concentrations. Airborne lead should no longer be a source of community exposure in the United States, but individual counties sometimes still exceed airborne lead regulations, and continued vigilance is warranted. Individual children may still be exposed to airborne lead in fumes or respirable dust resulting from sanding or heating old paint, burning or melting automobile batteries, or melting lead for use in a hobby or craft.

SOURCES OF LEAD EXPOSURE

Lead Paint, Dust, and Soil

The source of most lead poisoning in children now is dust and chips from deteriorating lead paint on interior surfaces.[13] Children who developed lead encephalopathy with blood lead concentrations more than 100 μg/dL often had chips of lead paint visible on abdominal plain films. Children who live in homes with deteriorating lead paint, however, can achieve blood lead concentrations of 20 μg/dL or greater without frank pica.[14] The use of leaded paint on interior surfaces ceased in the United States by the mid-1970s. However, in 1998, of the 16.4 million US homes with ≥1 child younger than 6 years, 25% still had significant amounts of lead-contaminated deteriorated paint, dust, or adjacent bare soil ("lead hazard").[2] Dust and soil are also a final resting place for airborne lead from gasoline and dust from paint. Lead in dust and soil can recontaminate cleaned houses[15] and contribute to elevating blood lead concentrations in children who play on bare, contaminated soil.[16]

Transplacental Exposure and Lead in Human Milk

Lead crosses the placenta, and the blood lead concentration of the infant is similar to that of the mother.[17] The source of lead in the infant's blood seems to be a mixture of approximately two thirds dietary and one third skeletal lead, as shown by studies that exploited the differences in lead isotopes stored in the bones of women migrating from Europe to Australia.[18] Although lead appears in human milk, the concentration is closer to plasma lead and much lower than blood lead, so little is transferred. Because infant formula and other foods for infants also contain lead, women with commonly encountered blood lead concentrations who breastfeed their infants expose them to slightly less lead than if they do not breastfeed.[19] In Mexico, giving women supplemental calcium during lactation resulted in a small (less than 2 μg/dL) decrease in the mother's blood lead concentration, presumably by decreasing skeletal resorption.[20] Theoretically, this could diminish transfer of lead through breast milk even further. In the United States, however, where calcium intake may be higher, calcium supplementation does not prevent bone loss during lactation[21] and, thus, might not affect lead transfer at all.

Other Sources

Lead plumbing (in Latin, "plumbus" = lead) has contaminated drinking water for centuries, and lead in water can contribute to elevated blood lead concentrations in children.[13] In 2003–2004, some tap water in Washington, DC, was found to exceed Environmental Protection Agency (EPA) regulations. This was thought to be caused by a change in water disinfection procedures, which increased the water's ability to leach lead from connector pipes between the water mains and interior plumbing in old houses. The extent of this problem in Washington and other cities is not yet known. Affected families are drinking filtered or bottled water until the pipes can be replaced. (Most bottled water is not fluoridated; its consumption may lead to marginal fluoride intakes in children.) Much more about lead in drinking water is available on the EPA Web site (www.epa.gov/safewater/lead/index.html).

Table 1 includes questions about less common sources of lead exposure, which include hobbies, contaminated work clothes, ceramics, cosmetics, imported canned foods, etc. Such questions may be useful if a child has an elevated blood lead concentration but no exposure to leaded dust or soil. They have not been validated for the purpose of deciding whether to screen.

The lead concentration of blood for transfusion is not routinely measured. After exchange transfusion in the extremely low birth weight infant, 90% of the infant's blood is donor blood. Bearer et al[22] recommended that only units with lead concentrations of less than 0.09 μmol/L be used in these patients, on the basis of their adaptation of the World Health Organization tolerable weekly intake from ingestion to intravenous injection. Approximately one third of the units of blood that they measured were above this concentration. The effect of lead in transfused blood used in older children has not been considered.

TOXICITY OF LEAD

Subclinical Effects

At the levels of lead exposure now seen in the United States, subclinical effects on the central nervous system (CNS) are the most common effects. The best-studied effect is cognitive impairment, measured by IQ tests. The strength of this association and its time course have been observed to be similar in multiple studies in several countries.[23] In most countries, including the United States, blood lead concentrations peak at approximately 2 years of age and then decrease without intervention. Blood lead concentration is associated with lower IQ scores as IQ becomes testable reliably, which is at approximately 5 years of age.[23] The strength of the association is similar from study to study; as blood lead concentrations increase by 10 μg/dL, the IQ at 5 years of age and later decreases by 2 to 3 points. Canfield et al[7] recently extended the relationship between blood lead concentration and IQ to blood lead concentrations less than 10 μg/dL. They observed a decrease in IQ of more than 7 points over the first 10 μg/dL of

TABLE 2. Summary of Recommendations for Children With Confirmed (Venous) Elevated Blood Lead Concentrations[16]

Blood Lead Concentration	Recommendations
10–14 μg/dL	Lead education
	Dietary
	Environmental
	Follow-up blood lead monitoring
15–19 μg/dL	Lead education
	Dietary
	Environmental
	Follow-up blood lead monitoring
	Proceed according to actions for 20–44 μg/dL if
	A follow-up blood lead concentration is in this range at least 3 months after initial venous test; or
	Blood lead concentration increases
20–44 μg/dL	Lead education
	Dietary
	Environmental
	Follow-up blood lead monitoring
	Complete history and physical examination
	Lab work
	Hemoglobin or hematocrit
	Iron status
	Environmental investigation
	Lead hazard reduction
	Neurodevelopmental monitoring
	Abdominal radiography (if particulate lead ingestion is suspected) with bowel decontamination if indicated
45–69 μg/dL	Lead education
	Dietary
	Environmental
	Follow-up blood lead monitoring
	Complete history and physical examination
	Lab work
	Hemoglobin or hematocrit
	Iron status
	Free EP or ZPP
	Environmental investigation
	Lead hazard reduction
	Neurodevelopmental monitoring
	Abdominal radiography with bowel decontamination if indicated
	Chelation therapy
≥70 μg/dL	Hospitalize and commence chelation therapy
	Proceed according to actions for 45–69 μg/dL
Not Recommended at Any Blood Lead Concentration	
Searching for gingival lead lines	
Evaluation of renal function (except during chelation with EDTA)	
Testing of hair, teeth, or fingernails for lead	
Radiographic imaging of long bones	
X-ray fluorescence of long bones	

ZPP indicates zinc protoporphyrin.

blood lead that are of concern now, the test is obsolete for that use; however, EP measurement is still used clinically in managing children with higher blood lead concentrations.

Clinical Effects

Children with blood lead concentrations greater than 60 μg/dL may complain of headaches, abdominal pain, loss of appetite, and constipation and display clumsiness, agitation, and/or decreased activity and somnolence. These are premonitory symptoms of CNS involvement and may rapidly proceed to vomiting, stupor, and convulsions.[34] Symptomatic lead toxicity should be treated as an emergency. Although lead can cause clinically important colic, peripheral neuropathy, and chronic renal disease in adults with occupational exposures, these symptoms are rare in children.

Reversibility

In an influential 1994 study, 154 children who were 13 to 87 months old and had blood lead concentrations between 25 and 55 μg/dL were given chelation with ethylenediaminetetraacetic acid (EDTA) and therapeutic iron when clinically indicated and then followed for 6 months. Those whose blood lead concentrations decreased the most had improved cognitive test scores independent of whether they had been given iron or chelation therapy.[35] An Australian study[36] of 375 children with longer follow-up, however, found only small and inconsistent improvement in the IQs of children

cause they are at especially high risk of exposure or have symptoms suggestive of lead poisoning (diagnosis).

Screening

Between 1991 and 1997, both the AAP and CDC recommended universal screening, that is, that all children have their blood lead concentration measured, preferably when they are 1 and 2 years of age. Because the prevalence of elevated blood lead concentrations has decreased so much, a shift toward targeted screening has begun,[43] and the criteria for and implementation of targeted screening continues to develop. As of early 2005, the situation is as follows. All Medicaid-eligible children must be screened.[4] Medicaid will reimburse 2 screenings, one at 1 year of age and one at 2 years of age. Most children with elevated blood lead concentrations are Medicaid eligible, and most Medicaid-eligible children have not been screened.[4] The Advisory Committee on Childhood Lead Poisoning Prevention has proposed criteria by which a state could acquire an exemption from this requirement, and the proposal is under consideration in the Secretary of Health and Human Services' office. Until such exemptions are granted, both the CDC[4] and AAP support universal screening of Medicaid-eligible children. The thinking behind the availability of exemptions is not primarily to decrease the number of screenings performed but rather to increase it among groups in which increased lead absorption will be found. Children whose families participate in any assistance program but who, for whatever reason, are not eligible for Medicaid should also be screened.

For children not eligible for Medicaid, several states and some municipalities have developed targeted screening recommendations or policies using suggestions made by the CDC,[43] their own data, or some combination of the 2. All practitioners should determine if such recommendations are in place where they practice. Appropriate contacts at state and city health departments with CDC-funded programs are listed on the CDC Web site (www.cdc.gov/nceh/lead/grants/contacts/CLPPP%20Map.htm).

The approach to screening children who are not eligible for Medicaid and who live in areas in which health authorities have not made locale-specific recommendations is less clear. Although targeted screening may be desirable, well-validated tools with which to achieve it are not yet in place.[44] In the absence of policy, current recommendations support screening all children who are not enrolled in Medicaid and who live in areas in which local authorities have not issued specific guidance.

There are now many case reports of children who are recent immigrants, refugees, or international adoptees who have elevated (sometimes very elevated) blood lead concentrations.[45] Such children should be screened on arrival in the United States.

Diagnostic Testing

Some experienced clinicians measure the blood lead concentration in children with growth retardation, speech or language dysfunction, anemia, and attentional or behavioral disorders, especially if the parents have a specific interest in lead or in health effects from environmental chemicals. However, a persistent elevation of blood lead concentration into school age is unusual, even if peak blood lead concentration at 2 years of age was high and the child's housing has not been abated. This is probably because hand-to-mouth activity decreases and the child's body mass increases. Thus, a low blood lead concentration in a school-aged child does not rule out earlier lead poisoning. If the question of current lead poisoning arises, however, the only reliable way to make a diagnosis is with a blood lead measurement. Hair lead concentration gives no useful information and should not be performed.[46] Radiograph fluorescence measurement of lead in bone is available in a few research centers and has been used in children as young as 11 years with acceptable validity for research purposes,[47] but it has no clinical utility as yet.

MANAGEMENT OF CHILDREN WITH ELEVATED BLOOD LEAD CONCENTRATIONS

In 2002, the national Advisory Committee on Childhood Lead Poisoning Prevention published a monograph, "Managing Elevated Blood Lead Levels Among Young Children."[16] The goal of the monograph was to provide an evidence-based, standard approach to management usable throughout the United States. Anyone involved with the management of children with elevated blood lead concentrations needs access to it. This section is consistent with the monograph.

The management of children with elevated blood lead concentrations is determined primarily by how high the concentration is (Table 2). Children with concentrations less than 10 μg/dL are not currently considered to have excess lead exposure. Children with concentrations 10 μg/dL or greater should have their concentrations rechecked; if many children in a community have concentrations greater than 10 μg/dL, the situation requires investigation for some controllable source of lead exposure. Children who ever have a concentration greater than 20 μg/dL or persistently (for more than 3 months) have a concentration greater than 15 μg/dL require environmental and medical evaluation.

Residential Lead Exposure

Most children with elevated blood lead concentrations live in or regularly visit a home with deteriorating lead paint on interior surfaces. Some children eat paint chips, but pica is not necessary to achieve blood lead concentrations of 20 μg/dL or greater.[14] Children can ingest lead-laden dust through normal mouthing behaviors by simply placing their hand or an object in their mouth. This also happens when children handle food during eating.[48–50] There is increasing evidence that professional cleaning, paint stabilization, and removal and replacement of building components can interrupt exposure. Cooperation with the health department in investigating and decreasing the source is necessary. Although some authorities insist that moving children to unleaded

typically given the drug, body surface area calculations give higher doses, which are those that are recommended.[54]

Although chelation therapy for children with blood lead concentrations of 20 to 44 μg/dL can be expected to lower blood lead concentrations, it does not reverse or diminish cognitive impairment or other behavioral or neuropsychologic effects of lead.[3] There are no data supporting the use of succimer in children whose blood lead concentrations are less than 45 μg/dL if the goal is to improve cognitive test scores.

Children with symptoms of lead poisoning, with blood lead concentrations higher than 70 μg/dL, or who are allergic or react to succimer will need parenteral therapy with EDTA and hospitalization. Guidelines for these circumstances are beyond the scope of this statement, but the same consultation as described above is recommended. There are academic centers that use D-penicillamine, another oral chelator used in Wilson disease, for lead poisoning. Its safety and efficacy, however, have not been established,[55] and the AAP Committee on Drugs considers it to be a third-line drug for lead poisoning.[56]

Dietary Intervention

The Advisory Committee on Childhood Lead Poisoning Prevention reviewed the evidence for dietary intervention in lead-exposed children.[16] They concluded that there are no trial data supporting dietary interventions aimed specifically at preventing lead absorption or modulating the effects of lead. However, there are laboratory and clinical data suggesting that adequate intake of iron, calcium, and vitamin C are especially important for these children. Adequate iron and calcium stores may decrease lead absorption, and vitamin C may increase renal excretion. Although there is epidemiologic evidence that diets higher in fat and total calories are associated with higher blood lead concentrations at 1 year of age,[57] the absence of trial data showing benefits and the caloric requirements of children at this age preclude recommending low-fat diets for them.

Psychological Assessment

The Advisory Committee on Childhood Lead Poisoning Prevention reviewed the evidence for psychological assessment and intervention in lead-exposed children.[16] Despite data from several large epidemiologic studies suggesting that moderate exposure to lead produces specific deficits in attention or executive functions, visual-spatial skills, fine-motor coordination, balance, and social-behavioral modulation,[58] there is no specific "signature" syndrome yet identified. In addition, although 2-year-olds tend to have the highest blood lead concentrations, they will usually not have detectable cognitive damage, which can be expected to become more apparent at 4 years of age and later. It seems reasonable to manage children whose blood lead concentration is 20 μg/dL or greater at its peak as having a higher risk of developmental delay and behavior abnormalities.[16] Because the effects emerge later, after the child's blood lead concentration will have decreased, the child's record must be kept open even after the blood lead concentration has decreased.

Although there is not specific literature supporting the use of enrichment programs in lead-poisoned children, programs aimed at children with delay from another cause should be effective in lead-poisoned children.

RECOMMENDATIONS FOR PEDIATRICIANS

1. Provide anticipatory guidance to parents of all infants and toddlers about preventing lead poisoning in their children. In particular, parents of children 6 months to 3 years of age should be made aware of normal mouthing behavior and should ascertain whether their homes, work, or hobbies present a lead hazard to their toddler. Inform parents that lead can be invisibly present in dust and can be ingested by children when they put hands and toys in their mouths.
2. Inquire about lead hazards in housing and child care settings, as is done for fire and safety hazards or allergens. If suspicion arises about the existence of a lead hazard, the child's home should be inspected. Generally, health departments are capable of inspecting housing for lead hazards. Expert training is needed for safe repair of lead hazards, and pediatricians should discourage families from undertaking repairs on their own. Children should be kept away from remediation activities, and the house should be tested for lead content before the child returns.
3. Know state Medicaid regulations and measure blood lead concentration in Medicaid-eligible children. If Medicaid-eligible children are a significant part of a pediatrician's practice or if a pediatrician has an interest in lead poisoning, he or she should consider participating in any deliberations at the state and local levels concerning an exemption from the universal screening requirement.
4. Find out if there is relevant guidance from the city or state health department about screening children not eligible for Medicaid. If there is none, consider screening all children. Children should be tested at least once when they are 2 years of age or, ideally, twice, at 1 and 2 years of age, unless lead exposure can be confidently excluded. Pediatricians should recognize that measuring blood lead concentration only at 2 years of age, when blood lead concentration usually peaks, may be too late to prevent peak exposure. Earlier screening, usually at 1 year of age, should be considered where exposure is likely. A low blood concentration in a 1-year-old, however, does not preclude elevation later, so the test should be repeated at 2 years of age. Managed health care organizations and third-party payers should fully cover the costs of screening and follow-up. Local practitioners should work with state, county, or local health authorities to develop sensitive, customized questions appropriate to the housing and hazards encountered locally.
5. Be aware of any special risk groups that are prevalent locally, such as immigrants, foreign-born

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Passing in the vicinity of Pittsburgh's new highway, June 1940.

other men would swarm across a field, far from the plant and its explosives, into wooden smoking shacks with glowing cigar lighters embedded in the walls. There, Needleman would smoke and check out his coworkers. In the corner, a few older men sat staring blankly into space, moving slowly and clumsily. If they spoke, their voices were distant and empty. One day, when Needleman asked other workers the story behind these men, they all shook their heads. "Oh yeah," one told him, "those guys worked in the House of Butterflies."

Needleman joined the University of Pittsburgh School of Medicine in 1981, leaving Harvard University to join Pitt's Departments of Psychiatry and Pediatrics. Calling professor Needleman a leader in the field of lead research would be an understatement. (The champion of preventive medicine has long since kicked the smoking habit, by the way.) He has spent much of his career attempting to convince others that exposure to lead, even at low doses, has tragic effects on individuals and society. Though few deny that high doses of lead are toxic, its low-dose effects have been passionately debated. If you ask Needleman where arguments against the danger of low-dose lead exposure come from, he'll tell you it's the lead industry—an entity he has fought through several turbulent decades. The battle starts with Needleman's first academic paper, and spans through scientific misconduct charges brought against him (by researchers who served as paid expert witnesses for the lead industry), to his work today.

As for the scientific misconduct charges, the committee that investigated him regarding the allegations directed Needleman to correct and clarify published reports of certain methodological aspects of his work and to make available to any interested scholars his complete data set on his tested subjects. More importantly, the committee asserted that the conclusions from his data were robust. Needleman had not engaged in scientific misconduct. Further, his early findings on subclinical lead exposure have since been confirmed by similar studies in Australia and elsewhere. And his efforts to de-lead America in the name of public health, even in the face of scalding controversy, have won him prestigious honors such as the Dana and Heinz Awards.

As for halting the effects of low-level lead exposure, Needleman has had a few victories, but at 73, it's a fight that still consumes him.

Rubbing his eyes gently, Needleman lets out a deep sigh when he talks about lead and its effects. "Lead does so many things to human biology, we don't even know which ones are most important," he says. It affects neurotransmitters responsible for nerve conduction, causes leaky capillaries, kills brain cells, affects RNA transferase and transcription of the genome, and that's just an abbreviated list. "There are thousands of articles out there," he says, "and so many effects that could be critical, we don't really know what's what," and then he pauses. "We just know that the more you look for brain effects, the more you find them, even at very low doses."

Needleman recalls how in 1960, according to the Centers for Disease Control and Prevention, a child needed at least 60 micrograms of lead per 100 milliliters of blood to be officially identified as poisoned. Back then, 20 percent of inner city children had blood lead levels of 40 to 50 micrograms per 100 milliliters, and they were considered normal. This made no sense to Needleman. *Listen,* he said, *if we know for a fact that high-dose lead poisoning causes obvious problems—like coma, retardation, and death—why should we assume that lower levels cause no injury to a child's brain?* He has asked this question repeatedly for about five decades. Almost every time he does, he designs a study to examine it from a new angle. (Today, the toxic lead level is defined as 10 micrograms per 100 milliliters, and still 21 percent of inner-city children have lead levels above that, according to Needleman.)

In the '70s, Needleman's community mental health office was what used to be a living room in an old brownstone in an impoverished section of Philadelphia. Each morning Needleman stared through his office window into a primary schoolyard across the street. It was full of poor kids, mostly minorities, who lived in turn-of-the-century houses with peeling lead paint. As they giggled and ran by his window, Needleman started to think to himself, *How many of those kids aren't going to make it because they are lead poisoned? And what other damage might they suffer from lead's toxins?* To find out, first he needed a better measuring stick. Lead is a bone-seeker—like calcium, it migrates into bone, where it accumulates. So if a child were exposed to lead during, say, the first three years of life, a blood-level test at four might not show any lead. At the time, the only accurate test of long-term lead exposure was a bone biopsy, which would not have been acceptable for hundreds of seemingly healthy children. But when a child loses a tooth, Needleman realized, it's like a spontaneous, pain-free biopsy. He got a $500 grant from the federal government, took a chunk of it to the local bank, and converted it into silver Kennedy half-dollars. Then he had little badges made up that said "I gave." With his half-dollars and badges, Needleman worked with the schools to collect teeth from several locations—some from Philadelphia's "lead belt" on North Broad Street, a hot spot for poisoning, others from areas that rarely reported lead poisoning. Those teeth, Needleman established, were good markers for lead levels.

That got him an invitation to Harvard, where he would show the world lead's subtle destructive powers. In 1979, in a study on Massachusetts children, he determined their life-long accumulation of lead and examined whether that correlated with their IQs. He found that children with higher accumulations of lead also had, on average, five or six fewer IQ points than those from the same neighborhood, ethnic background, and economic status with lower accumulations.

"That study," says Philip Landrigan, professor and chair of community and preventive medicine at Mt. Sinai School of Medicine, in New York, "really changed the whole way the world thinks about lead poisoning."

"He really made the world consider the possibility that subclinical exposure to environmental pollutants could have a serious societal impact," notes David Bellinger of Harvard Medical School, who has collaborated on studies with Needleman.

"These low-level exposures may not result in a child who is clinically ill, but he showed that there is a more subtle impact: It reduces the child's quality of life, and when the effect of lead is projected across the whole population, it has a cumulative impact that's really substantial. It's shifting the whole distribution of cognitive level a bit toward the lower end." Needleman calls this the subtle dumbing down of America; he doesn't take it lightly.

When people hear the story of Needleman working at Deep Water and seeing lead-poisoned workers from the House of Butterflies, they are likely to say, *Oh, that explains why he's anti-lead.*

But actually, it doesn't. For Needleman, the significance of that day at Deep Water did not hit him until years later, after an experience with a young Hispanic girl changed his understanding of lead poisoning and its causes.

It was the early 1960s, Needleman was a self-proclaimed "cocky" resident at Children's Hospital of Philadelphia, and a young girl, we'll call her Vanessa, was admitted to his ward with severe lead poisoning. She had eaten the lead-based paint peeling from her inner-city home, and her story was all too common. Her brain had swollen to a point where she was dangerously near death. She didn't cry, didn't smile, just lay there, comatose. Needleman treated her with EDTA, a chelating agent and the only drug available to counter lead poisoning. Soon, she woke up crying, and Needleman breathed a small sigh of relief. Within a few days, she smiled the sweetest smile Needleman can remember. He felt proud, even smug. When he knew the girl was going to make it, he turned to her mother and calmly told her she had to move from her home.

"If Vanessa eats more paint," he said, "there's no question she'll be brain damaged."

Her mother shot Needleman an angry look and snapped, "Where can I go? Any house I can afford will be no different from the house I live in now."

Needleman's smugness vanished. "I realized," he says, "that it wasn't enough to make a diagnosis and prescribe medication. I'd treated her for lead poisoning, but that was not the disease—the disease was much bigger and caused by forces embedded in the child's life. Her disease was where she lived and why she was allowed to live there."

Historically, childhood lead poisoning has been a problem for minorities and low-income families. "There's much more lead in poor, black, and Hispanic neighborhoods because of the kinds of houses they live in," Needleman points out. "There are middle-class white kids who are affected, but the rate is five to six times higher in the poor neighborhoods." Today, old paint is the most important factor, but for several decades, lead in gasoline compounded the problem. After the deaths at Deep Water and other plants, there was a brief moratorium on leaded gasoline. Soon after though, lead became a major component of everyday life in America, most notably as an additive to gasoline and paint. In 1973 alone, as Needleman puts it, "200,000 tons of lead were blowing out of the exhausts of American cars each year." He thought this was a crime. The more studies he conducted, the more deleterious effects from lead he found. Through governmental committees, editorials, and other means, Needleman and other researchers fought against leaded gasoline for 40 years.

"Dr. Needleman was a key figure in persuading the Environmental Protection Agency to take lead out of gasoline," says Landrigan. "That single action of taking lead out of gasoline has brought a 90 percent reduction in blood lead levels in children of this country."

Needleman wants to do the same for leaded paint. He says, "See, if you de-lead a house, that house is safe forever. It's not just the kid who's living there you're protecting—it's any kid who moves in. And in the poor neighborhoods, during the lifetime of that house, there may be 10 different families in there, so you're protecting all those children." Then he pauses.

"Dr. Needleman was a key figure in persuading the Environmental Protection Agency to take lead out of gasoline," says Landrigan. "That single action of taking lead out of gasoline has brought a 90 percent reduction in blood lead levels in children of this country."

"In a way," he whispers, "it's a bargain.

"People say we can't afford to do it. We can't afford not to do it. The actual cost-benefit analysis done by the Public Health Service shows that, in terms of avoided health costs and special education fees, there will be a $28 billion savings for de-leading all the houses. So there are a lot of good reasons to do it: moral, ethical, and practical reasons."

When moral and ethical motivations are involved, it seems Needleman will go to any lengths to right a situation, and it's not unlikely for him to upset a few people along the way. As an antiwar activist during the Vietnam War, for example, he traveled overseas to rescue wounded Vietnamese children and bring them to the United States for medical care. He and Benjamin Spock, the famous pediatrician who was a mentor for Needleman, spent their share of time together, including one night in jail for an antiwar protest. During all of this, Needleman kept up his fight against lead.

While at Harvard, Needleman studied newborns, taking blood from umbilical cords to determine prenatal lead exposure. He found that even at very low doses, infants born with higher lead levels had slower neurobehavioral development than those from the same backgrounds with less exposure in the womb.

Later, at Pitt, Needleman and his colleagues reexamined kids from the famous Harvard IQ study that he had conducted 11 years earlier. Those kids, at 17- or 18-years-old, were more likely to be dyslexic, drop or flunk out of high school, and get arrested if their lead levels surpassed 10 micrograms.

Most of the lead studies to date, including Needleman's, have focused on IQ, but he doesn't think that's the most important factor. "I think lead affects attention, behavior, and impulsivity," he says, quickly pointing out that this isn't a new idea. Another mentor, Randolph Byers at Children's Hospital in Boston, first saw this connection in a few patients referred to him for aggressive behavior during the '40s. But Needleman is the first to explore this connection through in-depth studies.

In 1996, Needleman conducted his first delinquency study; it involved several hundred children. He measured their bone lead levels and collected reports of aggression and delinquency from the subjects, their parents, and their teachers. With this study, Needleman showed an association between lead and delinquency. For him, the next logical step was to see if lead affected arrest rates. He identified about 200 adolescents who'd been sentenced to time behind bars and a control group of teens from local high schools with no arrest records. He measured the lead stored in their bone, using a relatively new non-invasive technique called X-ray fluorescence spectroscopy, and found that, controlling for race and socioeconomic class, mean lead levels in delinquents were significantly higher.

"Well," he says with a tisk, "that's a lot of delinquency. And the thing about lead toxicity is, it's completely preventable." He shakes his head. "Of all the causes of delinquent behavior, this is probably the easiest one to get at. If you just take lead out of the houses, then people won't get poisoned, and a significant amount of delinquency might well disappear. Just think of what that would do for our society."

"Lead, as Herb has said so many times, is a simple problem," says Bellinger. "We know where it is, how it gets into the body, and the damage it can do. In some ways, it's a bellwether of our abilities as a society to address these problems." ■

MASSACHUSETS DEPARTMENT OF PUBLIC HEALTH
EARLY INTERVENTION
ELIGIBILITY CRITERIA DEFINITIONS

CHILD CHARACTERISTICS

NOTE – Factors 1 – 4 apply only to children under 18 months chronological age. Birth or medical records are available to substantiate factors 1 – 4.

1. **Birth weight** – A child meets this risk criterion if the birth weight is less than 1200 grams (2 pounds 10 ½ ounces).

2. **Gestational Age** – A child meets this risk criterion if the gestational age of the child is less than 32 weeks.

   NOTE: Developmental evaluation for eligibility will be based on chronological age, not on adjusted age.

3. **NICU Admission** – This risk criterion applies to a child with a stay in the Neonatal Intensive Care Unit of more than 5 days.

4. **Apgar** – A child meets this risk criterion if the child's Apgar score was **less** than 5 at 5 minutes.

5. **Total Hospital Stay** –
   A child meets this risk criterion if the total number of days as an inpatient in a hospital or extended care facility exceeds 25 days in a 6-month period.

   NOTE: This does not apply to the birth admission of a premature child. Subsequent admissions to a hospital or the transfer hospital stay after NICU admission will apply toward this total.

6. **Intrauterine Growth Retardation/Small for Gestational Age** – A child meets this risk criterion if diagnosed at birth with Intrauterine Growth Retardation (IUGR) or Small for Gestational Age (SGA).

7. **Weight for Age and Weight for Height** –
   a. A child meets this risk criterion when **weight for age** or **weight for height** is less than the 5th percentile or greater than the 95th percentile.
   b. A child meets this risk criterion if the **weight for age** has **dropped** 2 or more major centiles in 3 months if child is under 12 months of age or has dropped 2 or more major centiles in six months if 12 to 36 months of age. A major centile is defined as the major percentiles (5, 10, 25, 50, 75, 90, 95) on the Physical Growth Chart adopted by the National Center for Health Statistics.

- Persistence of multiple signs of less than optimal sensory and motor patterns, including under-reaction or over-reaction to auditory, visual, or tactile input.

12. Multiple Trauma/Losses –

a. A child meets this risk criterion if he/she has experienced a series of traumas or extreme losses that may impact on the care and/or development of the child. For example, multiple hospitalizations or multiple placements outside the home.
b. This risk factor should be documented in the child's record and appropriate outcomes and treatment strategies addressed as determined by the family.

FAMILY CHARACTERISTICS

NOTE #1 – Regarding children in the care of someone other than the child's biological parent: If the DSS (Department of Social Services) goal is for the reunification of the parent and child, the following risk factors apply based on the biological parent. The EI program should work closely with both the biological and foster families of the child, whenever possible. If there is no goal for reunification with the child's biological parents, the family risk factors are to be based on the family characteristics of the primary caregivers.

NOTE #2 – Determination of risk factors under family characteristics should be determined by family perception.

NOTE #3 – Maternal characteristics apply as risk factors to fathers if the father is the primary caregiver.

1. **Maternal Age/Parity –**
   a. A mother meets this risk criterion if her age at the time of the child's birth was less than 17 years.
   b. A mother meets this risk criterion if she has given birth to 3 or more children before the age of 20.

2. **Maternal Education** – A mother meets this risk criterion if she has completed 10 years or less of formal education at the time of the eligibility evaluation.

3. **Parental Chronic Illness or Disability –**
   a. A family meets this risk criterion if one parent has a diagnosed chronic illness or a sensory, mental, or developmental disability which is likely to interfere with or adversely affect the child's development or have an impact on care-giving abilities.
   b. Examples of this risk factor may be affective disorders, schizophrenia, sensory limitations, including visual or hearing limitations, and cognitive limitations.

Connecticut Birth to Three System

BIRTH to THREE SUPPORTS

DIAGNOSED CONDITIONS LIST - Automatic eligibility

These diagnoses have a high probability of resulting in developmental delay even if no delays currently exist, and therefore entitle children to Birth to Three supports when documented by a physician (or an audiologist in the case of hearing impairment).

Genetic Disorders
- A. Chromosomal Abnormality Syndromes (758._)
  - All (except Klinefelter Syndrome)
- B. Pre-natal exposures
  - Fetal Alcohol Syndrome (760.71)
  - Fetal Phenytoin (Dilantin) Syndrome (760.79)
- C. Neurocutaneous Syndromes
  - Tuberous Sclerosis (759.5)
- D. Inborn Errors of Metabolism
  - i. Amino Acidopathies
    - Organic Acidemias (270.3)
    - Glutaric Aciduria type II (270.9)
  - ii. Very long chain fatty acid storage diseases (330.9)
    - All, includes Peroxisomal Disorders (330)
- E. Pre-natal Infections
  - TORCH:
    - congenital toxoplasmosis (771.2)
    - congenital rubella (771.0)
    - congenital CMV (cytomegalovirus) (771.1)
    - congenital herpes (771.2)
- F. Other Syndromes
  - Angelman Syndrome (759.89)
  - Bardet-Biedl Syndrome (759.89)
  - CHARGE Syndrome (759.89)
  - Cornelia de Lange Syndrome (759.8)
  - Jeune Syndrome (756.4)
  - Lissencephaly Syndrome/Miller-Dieker Syndrome (742.2)
  - Merikes Syndrome (759.51)
  - Noonan Syndrome (759.89)
  - Opitz Syndrome (759.89)
  - Prader-Willi Syndrome (759.81)
  - Rubenstein-Taybi Syndrome (759.89)
  - Weaver Syndrome (759.89)
  - Williams Syndrome (759.89)

Sensory Impairments
- Congenital or acquired
- Not unilateral
- Auditory Neuropathy (389.9)
- Blindness ("legal" blindness or 20/200 best achievable acuity with correction) (369._ _)
- Low vision (20/70 best acuity with correction (369._ _)
- Retinopathy of Prematurity, grade 4 or grade 5 (362.21)
- Hearing Impairment (40dB loss or greater) (389._ _)

Motor Impairments
- Developmental Apraxia of Speech (784.69)

Neurologic Disorders
- Brain Malformation (742.9)
- Cerebral Dysgenesis (742.2)
- Cerebral Palsy (all types) (343._)
- Degenerative Progressive Neurological Condition (330.9)
- Encephalopathy (742.2)
- Holoprosencephaly (742.2)
- Hydrocephaly, congenital (742.3), or acquired (331.4)
- Intraventricular Hemorrhage (IVH) - grade 3 or grade 4 (772.1)
- Meningomyelocele / Myelomeningocele / Spina Bifida / Neural Tube Defect (741.9)
- Myopathy (359.81)
- Peri-ventricular Leukomalacia (PVL) (742.4)
- Porencephalic Cyst (742.4)
- Seizures (poorly or uncontrolled) (345.9)
- Spinal Muscular Atrophy / Werdnig Hoffman Disorder (335.0)
- Stroke (436)

Sociocommunicative Disorders
- Asperger Syndrome / Disorder (299.0)
- Autism (299.0)
- Childhood Depression (311)
- Childhood Disintegrative Disorder (299.1)
- PDD-NOS (299)
- Reactive Attachment Disorder (315.8)
- Rett Syndrome (330.8)

Medically Related Disorders
- Congenital or infancy-onset hypothyroidism (243)
- Cleft Palate (prior to the operation to repair the cleft and up to one year post-operative) (749.0)
- Lead Intoxication (> 45 µg/dL) (up to six months after identification) (984.9)
- Very Low Birth Weight (<750 grams at birth) (765.1 if under 500g or 765.2 if 500g-749g) - up to 6 months corrected age *only*

Acquired Trauma Related Disorders
- Traumatic Brain Injury / TBI (854)

Disorders of Growth
- None

FOLLOW-ALONG VISITS

Children found NOT eligible for Birth to Three will be offered free quarterly follow-along visits when they have:

(1) a birth weight between 750g - 999g (ICD-9 code 765.3) when evaluated prior to 6 months correct age, or

(2) at least 2 SD below the mean in expressive language only plus a biological risk factor, or

(3) a condition listed below along with 1.5 SD below the mean in at least one area of development

Genetic Disorders
- A. Chromosomal Abnormality Syndromes
  - Klinefelter Syndrome (758.7)
- B. Pre-natal exposures
  - Fetal Alcohol Effects - not syndrome (760.79)
- C. Neurocutaneous Syndromes
  - Sturge-Webber Syndrome (759.6)
- D. Inborn Errors of Metabolism
  - Urea Cycle Defects and Hyperammonemias (270.6)
  - Amino Acidopathies (270)
- E. Other Syndromes
  - Achondroplasia (dwarfism) (756.4)
  - Apert Syndrome (755.55)
  - DiGeorge Syndrome (279.11)
  - Goldenhar Syndrome / Hemifacial Microsomia / Oculo-auriculo Vertebral Abnormality (756.0)
  - Moebius Syndrome (352.6)
  - Osteogenesis Imperfecta - types 2 & 3 (756.51)
  - Pfeiffer Syndrome (755.55)
  - Pierre-Robin Syndrome (756.0)
  - Russell Silver Syndrome (759.89)
  - Treacher Collins Syndrome (756.0)
  - VATER Association (759.89)

Sensory Impairments
- Chronic Otitis Media (for more than six months) (382.9)

Motor Impairments
- Arthrogryposis / Multiplex Congentia (754.89)
- Severe Scoliosis (754.2)

Neurologic Disorders
- Central Congenital Hypoventilation Syndrome (306.1)
- Microcephaly (742.1)
- s/p Encephalitis (323.9)
- s/p Meningitis (310.8)

Sociocommunicative Disorders
- None

Medically Related Disorders
- Cleft Palate (more than one year after the repair of the cleft. See Service Guideline #3) (749.0)
- Lead Poisoning (20 - 45 µg/dL) (up to six months after identification) (984.9)

Acquired Trauma Related Disorders
- None

Disorders of Growth
- "Failure to Thrive" (783.4)